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Intracranial Lesions on Diffusion weighted Imaging-A Case Series

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Original Research Article

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Abstract: This study was conducted to study the features of intracranial lesions of Diffusion weighted MR imaging and their correlation on other MRI sequences, to evaluate the role of DWI in etiopathogenesis and prognosis of intracranial lesions. A prospective, observational and cross section study was performed of 100 patients in 2 years period. Patient with clinical suspicion of stroke, CNS infections, head injury, demyelination and tumor were included. MRI's were done on SIEMENS Magnetom Avanto 1.5 Tesla. Age wise distribution, complaints, intra and extra axial location of tumor, presence and absence of restricted diffusion and findings of various sequences of MRI were studied. Maximum cases were observed from 4th - 6th decades.63% of cases were observed in males while 37 of cases were seen in females.93 cases were intra-axial, 7 were extraaxial. 80% of cases showed true restriction while 20% cases showed absence of restriction. Blooming on GRE was noted in 19% of cases.56% cases of stroke comprised of arterial infarct (39%), venous infarct (3%), and white matter ischemia (9%), and acute laminar necrosis (1%), hypoxic ischemic encephalopathy, PRES (1%) and parenchyma hemorrhage (1%).15% of cases were tumor which included glioma, lymphoma, metastasis, PNET and GBM Recurrence, 5 cases of traumatic brain injury (DAI), 9 cases of epidermoid cyst (5%) and cavernous haemangioma (5%) and 6 cases of brain infection were included. MRI features of these lesions on conventional MRI sequence, DWI and ADC maps were studied. All acute and subacute arterial infarcts showed restriction on diffusion weighted images and chronic cases did not show restriction. And all three cases of venous infarcts showed restriction diffusion due to cytotoxic edema. High grade gliomas, astrocytoma's, PNETS, lymphoma and metastasis showed diffusion restriction. Low grade gliomas did not show restriction. Keywords: Diffusion weighted Imaging (DWI), Apparent Diffusion Coefficient (ADC), Infarct, Brain Abscesses, brain tumors, Demyelination, diffuse axonal injury, encephalitis.

INTRODUCTION

DW imaging is a mathematical combination of T2W and DW images. Pure diffusion weighted image is obtained by subtracting the T2W image from the DW image. The resultant mapping of DW values in a voxel is called ADC mapping. ADC mapping represents true diffusion without T2 influence. Signal intensity on a gray scale is directly related to ADC values on DWI. Brain tissues with low ADC appears relatively hypointense, whereas regions with higher ADC values appears hyperintense. ADC is a value that describes microscopic water diffusibility in the presence of factors that restrict diffusion within tissues. In tissues, DWI probes the movement of water molecules, which occurs largely in the extracellular space. However, the movement of water molecules in the extracellular space is not entirely free, but is modified by interactions with hydrophobic cellular membranes and macromolecules. Hence, diffusion in biological tissue is often referred to as "apparent diffusion". By comparing differences in the apparent diffusion between tissues, tissue characterization becomes possible. For example, a

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tumor would exhibit more restricted apparent diffusion compared with a cyst because intact cellular membranes in a tumor would hinder the free movement of water molecules.

ADC values are calculated automatically by the software and then displayed as a parametric map that reflects the degree of diffusion of water molecules through different tissues. Then, by use of a dedicated workstation, ADC measurements are recorded for a given region by drawing regions of interest (ROIs) on the ADC map [1]. An ADC of a tissue is expressed in units of mm2/s. There is no unanimity regarding the boundaries of the range of normal diffusion, but ADC values less than 1.0 to 1.1 x 10-3 mm/s (or 1000-1100 x 10-6 mm2/s) are generally acknowledged in adults as indicating restriction. However, this is entirely dependent on the organ being imaged and the pathology[2]. The 'A' stands for apparent because we do not measure the pure diffusion coefficient (D or DC). In living tissue the diffusion process is superimposed by capillary pseudo diffusion and gross motion to which the MR measurement is also very sensitive. The Diffusion image (b 1000) displays reduced diffusion as hyperintense (brighter pixels); in contrast the ADC map displays it as hypointense (darker pixels).

Because DW MR imaging uses fast (echoplanar) imaging technology, it is highly resistant to patient motion, and imaging time ranges from a few seconds to 2 minutes. As a consequence, DW MR imaging has assumed an essential role in the detection of acute brain infarction and in the differentiation of acute infarction from other disease processes. DWI is useful in detection of acute stroke, demyelination, cerebral abscess and subdural empyema, encephalitis and celebrities, tumors, differentiation between Arachnoid cyst versus Epidermoid Cyst, diffuse axonal injuries and certain leucodystrophies [3].

This study was conducted to study the features of intracranial lesions of Diffusion weighted MR imaging and their correlation with other MRI sequences, to evaluate the role of DWI in etiopathogenesis and prognosis of intracranial lesions.

MATERIAL AND METHODS

Total 100 patients who underwent MR imaging of the brain during a period of 2 years were randomly considered for the study. These patients were prospectively subjected for DWI along with conventional MRI study. This was a prospective, observational and cross sectional study. The study was conducted on patients of age group (0 - 80 years), of either sex, referred to the Department of Radio diagnosis and Imaging, DR. D. Y. Patil Medical college, Pune. A written informed consent was taken for every patient before performing the MRI study. All studies were done on S IEMENS MAGNETOM AVANTO 1.5 Tesla MR System.

All patients clinically suspected of acute and chronic stroke, ischemia; intracerebral hemorrhage; Intracranial tumors (lymphoma, gliomas, metastasis); Extra axial masses (arachnoids cyst and epidermoid cyst, meningioma); Cerebral abscess; Encephalitis and celebrities; Head injury showing diffuse axonal injury; demyelination; sustained seizure activity; metabolic or toxic insults to the brain and leucodystrophy were included in this study. Patients having history of cardiac Pacemaker, cochlear implant, metallic foreign body metallic orthopedic hardware, pregnancy and severe claustrophobia were excluded. Diffusion tensor imaging will not be included in the study. Patient was placed in supine position and brain coil was used. MRI features of lesion detected were studied - site (intra or extra axial), margins, perilesional edema, appearance on T1WI, T2WI, FLAIR, GRE, presence of classification or haemorrhage, appearance on DWI and ADC values, any enhancement in contrast study. Non Ionic intra venous contrast OMNISCAN (gadodiamide) was administered in patients with normal renal function tests wherever necessary. The recommended dosage of OMNISCAN (gadodiamide) Injection is 0.2 mL/kg (0.1 mmol/ kg) administered intravenously. Though in general protocol, all the study specific sequences were taken i.e. FLAIR, Axial, Coronal, T1 Sagittal, T1 Axial, T2 Axial, GRE, and Diffusion weighted sequences, Apparent Diffusion coefficient- maps were obtained during post processing.

OBSERVATIONS AND RESULTS

Age (Yrs.)	No of cases	Percentage
1 - 10	6	6
11 - 20	6	6
21 - 30	7	7
31 - 40	9	9
41 - 50	22	22
51 - 60	22	22
61 – 70	23	23
>70	5	5

 Table-1: Age wise distribution of cases in study group

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Fig-1: Age wise distribution of cases in study group

The most common age group involved in our study was between 61-70 years followed by age group 41-50 years and 51-60 years.

In Our study there was a male predominance. 63 % were males and 37% were females (Fig-2).

Table-2. Sex v	vise distribution	of cases in	study groun
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Sex	No of cases	Percentage
Male	63	63
Female	37	37
Total	100	100



Fig-2: Sex wise distribution of cases in study group

Table-3: Complaints wise distribution of cases in study group

Complaints	No of cases	Percentage (n=100)
Headache & Vomitting	39	39
Weakness	35	35
Convulsion	38	38
RTA/fall	4	4
LBW/IUGR	4	4
Others	7	7



Fig-3: Complaints wise distribution of cases in study group

The most common presenting complaint/ symptom in our study were headache 39% and convulsion 38% followed by weakness 35%. Out of the 100 cases which we studied 93% were intra axial and 7% were extra axial in location (Fig-4).

Table-4: Intra and Extra axial location wise distribution of cases in study group

Location	No of cases	Percentage
Intra axial	93	93
Extra axial	7	7
Total	100	100



Fig-4: Intra and Extra axial location wise distribution of cases in study group

Table-5: Restricted diffusion wise distribution of cases in study group

Restricted diffusion	No of cases	Percentage
Present	80	80
Absent	20	20
Total	100	100



Fig-5: Restricted diffusion wise distribution of cases in study group

Out of 100 patients in our study 80% of our patients showed restriction of diffusion while 20% did not show restriction.

Out of 100 patients 81% showed blooming on FFE images while 19% did not show blooming (Fig-6).

Table-6: Blooming on GRE images wise distribution of cases in study group

Blooming on GRE images	No of cases	Percentage
Present	19	19
Absent	81	81
Total	100	100



Fig-6: Blooming on GRE images wise distribution of cases in study group

Table-7: Arterial ir	nfract wise o	distribution o	f cases in st	udy group

Arterial infract	No of cases	Percentage
Hemorrhagic	5	5
Non hemorrhagic	34	34
Total	39	100



Fig-7: Arterial infract wise distribution of cases in study group

Out of 34 patients 0f arterial infarct 12.82 % showed blooming on GRE (Hemorrhagic) images while 87.18 % did not show blooming (non hemorrhagic).

Out of 3 patients of venous infarct 66.67 % showed blooming on GRE (Hemorrhagic) images while 33.33% did not show blooming (non hemorrhagic) (Fig-8).

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Venous infract	No of cases	Percentage
Hemorrhagic	2	66.67
Non hemorrhagic	1	33.33
Total	3	100



Fig-8: Venous infract wise distribution of cases in study group

Table-9: Tumours showing restricted diffusion on diffusion weighted images in study group

<u> </u>		
Tumours	No of cases	Percentage
Restricted diffusion	11	73.33
Non restricted diffusion	4	26.67
Total	15	100



Fig-9: Tumours showing restricted diffusion on diffusion weighted images in study group

In our study out of the 15 tumor cases we studied 11 (73.33%) of the cases showed restricted diffusion while the 4 (26.67%) did not show restricted diffusion.

Out of the 6 Infective pathologies in our study, 5 (83.33%) of the cases had restriction of diffusion while 1(16.67%) of them did not show restriction (Fig-10).

Table-10: Infective etiology showing restricted diffusion on diffusion weighted images in study group

Infective	No of cases	Percentage
Restricted diffusion	5	83.33
Non restricted diffusion	1	16.67
Total	6	100



Fig-10: Infective etiology showing restricted diffusion on diffusion weighted images in study group

	MRI diagnosis	No of cases	Percentage
STROKE (Ischemia, Infarct, Hemorrhage) (n=56)	Arterial infarct	39	39
	Venous infarct	3	3
	White matter ischemia	9	9
	Acute cortical laminar necrosis	1	1
	Hypoxic Ischemic Encephalopathy	2	2
	Posterior reversible Encephalopathy		
	Syndrome	1	1
	Parenchymal Hemorrhage	1	1
Tumors (n=15)	Glioma/Astrocytoma	9	9
	Lymphoma	3	3
	Metastasis	1	1
	Premitive Neuroectodermal tumor	1	1
	Glioblastoma multiform recurrence		
		1	1
Trauma(n=5)	Diffuse axonal injury	5	5
Demyelination (n=4)	Multiple Sclerosis	1	1
	Tumefective Demyelination	1	1
	PML	1	1
	Leighs Disease	1	1
Epidermoid cyst(n=9)		9	9
Cavernous Haemangioma(n=5)		5	5
Infection (n=6)	Bacterial Parenchymal abscess	2	2
	Fungal Abscess	1	1
	Tuberculous Abscess	1	1
	Subdural Emphyma	1	1
	Viral rickettesial Encephalopathy	1	1
Total(n=100)		100	100

Table-11: MRI diagnosis wise distribution of cases in study group



DISCUSSION

According to our study gender distribution in intracranial lesions was males (63%) and females (37%) with the ratio of M: F being approximately 1.7:1. In our study the age distribution was from 0 to 80 years. Maximum cases were seen in 4th, 5th and 6th decade, by the age group between 51-70 years and 41-50 years. The youngest patient was a child of 6 months with global acute cortical laminar necrosis and the oldest patient was of the age group of 75 years with acute arterial territory infarct. In our study 39% patients present with headache and vomiting, 73% presented with focal neurodeficit like weakness (35%) and convulsions (38%).

Our study consisted of fifty six patients with stroke. Of these, 39 were of arterial infarct with 34 being non haemorrhagic infarcts and 5 being hemorrhagic. 3 were of venous infarcts of which 2 were non hemorrhagic and 1 was of haemorrhagic, 9 of white matter ischemia, 1 of acute cortical laminar necrosis, 2 HIE, 1 of PRES and 1 of parenchyma haemorrhage. Out of 42 cases of infarct, 37 were acute, 4 were subacute and 1 was chronic infarcts. Restriction was seen in acute and subacute and not seen in chronic cases (Figure 12). Interruption of blood flow through an intracranial artery leads to deprivation of oxygen and glucose in the supplied vascular territory. This initiates a cascade of events at a cellular level which, if circulation is not re-established in time, will lead to cell death, mostly through liquefactive necrosis. The mechanism of vessel obstruction is important in addressing therapeutic manoeuvres to both attempts to reverse or minimise the effects and to prevent future infarcts.

DWI is sensitive to restriction of Brownian motion of extracellular water due to imbalance caused by cytotoxic edema. Normally water protons have the ability to diffuse extracellular and loose signal. High intensity on DWI indicates restriction of the ability of water protons to diffuse extracellular. In humans, ischemic changes are detected with DWI as early as 2 to 6 hours after onset of symptomsarly. ADC pseudo normalization occurs in second week [4].



Fig-12: Acute non hemorrhagic infarct in left occipital lobe (PCA territory) appearing hyperintense on Axial FLAIR (A), showing restricted diffusion on DWI (B) with low ADC values(C)

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The pathogenesis of venous infarct is different from arterial ischemic infarction. During venous infarct, initially there is increase in venous pressure with disruption of capillary tight junction with increase in the volume of extracellular water (vasogenic edema). These lesions are completely reversible with successful venous thrombosis. Subsequently, there is increase in intracellular water volume (cytotoxic edema) with resultant restricted diffusion on DWI with low ADC values. The mechanism is energy failure with loss of Na+/ K+ pump activity as in arterial stroke. The swollen cells due to cytotoxic edema might be functionally but not irreversibly damaged, hence have a potential for recovery. Hence, venous infarcts with restricted diffusion due to cytotoxic edema are not associated with poor outcome after thrombolysis (Figure 13). The high signal intensity on DWI and low ADC value do not always indicate irreversibility, but rather the tissue at risk [5].



Fig-13: Venous hemorrhagic infarct in left frontal lobe due to superior sagittal sinus thrombosis appearing hyperintense on Coronal T1W images(A), showing blooming on GRE (B), restriction on DWI (C) and loss of signal on ADC (D) images. The 3D Veno image (E) reveals thrombosis of superior sagittal sinus.

Hypoxic ischemia encephalopathy limits the oxygen supply to areas of brain with high metabolic activity and high density of glutamate receptors. Patterns of HIE vary according to the time and duration of the insult. Diffusion weighted MR imaging is the earliest sequence to detect hypoxic ischemic encephalopathy due to early cytotoxic edema. There is restricted diffusion seen in affected areas of brain in HIE appering bright with reduced apparent diffusion coefficient on ADC maps (Figure 14).DWI sequences can detect HIE even when other sequences are normal or cublty abnormal. Restricted diffusion is an dynamic process beginning on day 1 of life and progress over next week. Dengue of restricted diffusion increases over the first few days of life, achieving maximum decrease of ADC values at approx. 5 days. Infact new regions of restricted diffusion not seen on day 1 may evolve in 1 day of life. ADC pseudonormalization occurs at the end of first week [6].



Fig-14: Axial T2 (A) and FLAIR (B) images show cystic encephalomalacic changes in frontal, parietal, occipital and temporal lobes on both sides involving grey and subcortical white matter. DWI images(C) show restriction in Genu (marked in blue), Splenium (marked in yellow) of corpus callosum suggestive of acute hypoxic (ischemic) injury.

Our case of PRES shows restriction on DWI, whereas normally DWI shows no restriction. Restricted diffusion is the second most common atypical presentation of PRES. This has a very important implication, as lesions with cytotoxic edema may progress to infarction. Several studies suggested the role of DWI in the prediction of development of infarctions in these cases. Other studies, however, suggested that PRES is reversible even with cytotoxic patterns (Figure 15).

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Fig-15: A case of Posterior Reversible encephalopathy Syndrome showing hyperintense lesions in bilateral cerebellar hemispheres (green arrow) on axial FLAIR (A) and T2WI (B) images , appearing hypointense on T1W images(C) and showing restriction of diffusion on DWI images (D).

White matter ischemic appears hyperintense on T2 and FLAIR, without restriction on DWI. Importantly the aetiology of PVWM and DWM changes differs. The latter is chronic small vessel ischaemic in nature, whereas the former (periventricular) relates to a combination of demyelination, ependymitis granularis, subependymal gliosis. Importantly they are not ischaemic in nature[7].

Mechanism of laminar cortical necrosis is not well understood. It represents cytotoxic edema affecting a particular layer of cerebral cortex, neuropathologically characterized by delayed selective neuronal necrosis and is consequence of HIE, hypoglycaemic encephalopathy, ischemic stroke and status epilepticus.It appears hyperintense on T1WI seen about 2 weeks after the ictus and becomes prominent at 1-2 months and began to fade at 3 months. T1 hyperintensity is due to accumulation of denatured proteins in dying cells and / or lipid taken microphages and do not occur due to haemorrhage or calcium. On T2WI, it appears hyperintense or isointense to unaffected cortex. On DWI it shows restricted diffusion with low ADC values [8, 9].

Parenchymal hemorrhage can pose some challenges in that the appearance of blood changes depending on the sequence and the time since the haemorrhage and the size and location of the bleed.

STAGE	T1WI	T2WI	DWI	ADC
HYPERACUTE	Iso	hyper	hyper	low
ACUTE	Iso	hypo	hypo	Cannot be calculated
EARLYACUTE	hyper	hypo	hypo	Cannot be calculated
LATEACUTE	hyper	hyper	hyper	hyper

For infarctions imaged within 24 hours, a study conducted in 1987 by Andrew Kertesz, Sandra E. Black, Larry Nicholson, and Thomas Carr on 175 patients reported a sensitivity of 58% for CT scan and 82% for MR Imaging [10].

Another study conducted by Hoehn-Berlage M in 1995 concluded that diffusion-weighted NMR imaging (DWI) has the potential to be a sensitive tool for the observation of stroke evolution [11]. Another study conducted in the year 1995 by Moseley ME *et al.* have demonstrated that changes in the ADC occur as early as 10 minutes following onset of ischemia which all correlated well with our study. Our results correlate well with the previous above mentioned studies [12].

A total of 15 cases of tumors were studied in this group. 11 patients showed restriction of diffusion with corresponding low values on ADC while 4 patients did not show restriction. High grade gliomas, astrocytoma's, PNETS, lymphoma and metastasis showed restriction diffusion (Figure 16). Rest was low grade gliomas showing no restriction. MR diffusion imaging has been used to study water mobility in normal brain tissue, cerebral infarction, multiple

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sclerosis, gliomas, and brain abscesses and to differentiate between arachnoid cysts and epidermoid cysts and other diseases. Gliomas are the most common brain tumors. On imaging studies, after intravenous contrast injection malignant gliomas usually shows enhancement with peritumoral edema. Differentiation of these two types of tumors occasionally may be difficult, because low-grade astrocytomas also may show abnormal contrast enhancement and peritumoral edema. Abnormal enhancement was seen in four of eight patients with grade II astrocytoma in our study, and peritumoral edema was found in four patients. We found that ADC values cannot be used in individual cases to differentiate tumor types reliably. Although the ADCs of grade II astrocytoma and glioblastoma overlapped somewhat, the combination of routine image interpretation and ADC had a higher predictive value. Our results indicate that lower ADCs suggest malignant glioma, whereas higher ADCs suggest lowgrade astrocytoma. These results agree with those of previous reports. No patients with anaplastic astrocytoma were included in our study; we expect that the ADCs of this type of tumor (a grade III astrocytoma) will be intermediate between those of glioblastoma and grade II astrocytoma. An ADC value of tumours is inversely correlated with cellularity. Hence, lymphomas, medullobastomas and PNETS have lower ADC values [13, 14].

DW Imaging for Grading of Glioma Tumors: Multiple studies have shown lower water diffusibility in high-grade gliomas than in lower grade gliomas. However there is considerable overlap between ADCs in high and low grade tumors making it difficult to use ADC as a biomarker to measure cellularity and hence grading. It remains doubtful whether tumor grade can be evaluated with enough specificity with DW imaging to be useful in a clinical context. At present DWI-ADC cannot be used to differentiate between high and lowgrade gliomas or between tumor types. In a study by Klisch J *et al.* in year 2000 done on three pediatric patients state that diffusion-weighted imaging (DWI) can be used to show the solid portion of the tumor preoperatively and to monitor postsurgical recovery[15].

In the present study, on ADC maps, most of the lesions were showing increased diffusion and in patients in whom histopathology reports were available was comparable with microcyst formation in these tissues. There was no significant difference between these individuals' subgroups of tumors e.g. gliomas versus meningioma's, gliomas vs. metastases and meningioma versus metastases based on findings of ADC maps. Our findings correlate well with previous studies done.



Fig-16: A case of cerebellar metastasis proved to be due to Ca Bronchus appearing hypointense on T1WI (A), heterogeneously hyperintense on T2WI (B), showing peripheral and heterogeneous enhancement in contrast study (C), showing restriction diffusion on DWI (D) with low ADC values (E). MRI chest (F)showing Ca Bronchus in right upper lobe (yellow arrow).

Bacteria, viruses, fungi, nematodes and parasites can all cause intracranial infections. By location they include epidural (extradural) abscess, subdural empyema, pachymeningitis, leptomeningitis, cerebritis, cerebral abscess, ventriculitis.In our study, a total of 6 cases of infections were studied. Out of these, 2 were bacterial parenchymal abscess, 1 was subdural emphyma, 1 was fungal abscess (Figure 17), 1 Viral Rickettesial Encephalopathy and 1 was tuberculous abscess. In this group all the patients showed restricted diffusion with low ADC values. In general all the lesions showed increased diffusion on ADC maps due to vasogenic edema. Presence of viscous fluid containing cellular debris, bacteria, inflammatory cells, and mucoid proteins causes restriction diffusion in abscesses is likely. Animal models have shown an inverse correlation between cellular density and ADC. And from the StokesEinstein equations diffusivity is inversely proportional to viscosity. As treated abscesses mature, central liquefaction occurs and T2"shinethrough" gradually replaces the ADC driven high signal on DW images [16,17]. In a study conducted in the year 1996 by Ebisu T et al stated that high signal intensity was observed in the abscess fluid, associated with low ADC [16].

In yet another study by Kim YJ *et al.* in 1998 on five patients with proven brain abscesses were prospectively evaluated with diffusion-weighted imaging and found that on diffusion-weighted imaging, brain abscesses showed markedly hyperintense signal in all five patients, and concluded that diffusion-weighted imaging may allow the differentiation of brain abscess from necrotic or cystic lesions[18]. Abscess cavities (Figure 18) and empyema's (Figure 19) are homogeneously hyperintense on DW MR images, with loss of signal intensities on ADC. Our findings correlate well with previous studies.

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Fig-17: A proven case of fungal granulomas, multiple supra and infra tentorial lesions were seen in bilateral fronto-parieto-ocipital region. These showed hyperintense core with hypointense rim on T2W images (A) with significant edema. Post contrast images showed peripheral rim enhancement (B).DWI Images (C) showed diffusion restriction with low ADC values (D).



Fig-18:A case of right frontal lobe abscess appearing hyperintense on T2W images with hypointense rim and extensive perilesional edema (A), showing peripheral rim enhancement in contrast (B) and showing restriction on DWI (C), with low ADC values (D).



Fig-19: A case of subdural empyema along right leaf of tentorium cerebeli with right sigmoid sinus thrombosis due to right mastoid CSOM appearing hyperintense along the inferior aspect of the tentorium cerebelli on the right (A), showing peripheral rim enhancment in contrast (yellow arrow) and filling defect in right sigmoid sinus (red arrow); showing restriction on DWI (C) with low ADC values (D).

A total of 4 patients of demyelinating diseases were studied under this group of which 1 was of Tumefactive Demyelination (Figure 20), 1 patient of Multiple sclerosis, 1 patient of Progressive Multifocal Leucoencephalopathy (Figure 21) and 1 patient of Leigh's disease(Figure 21). All the patients of demyelination showed restricted diffusion on DW images with low ADC values. My study correlated well with the study done previously.

Reduction of the motion along pathways reflects the interaction between the water molecules and the damaged structures, called "restricted diffusion." There are many causes for restricted diffusion in demyelinating diseases, including intramyelinic edema (cytotoxic oligodendroglia edema) or myelin vacuolation and reversible reduced vascular input. Myelin breakdown and inflammatory cell infiltration may reduce water movement in the extracellular space because of reduced fiber tract organization.

The presence of peripheral restricted diffusion in acute demyelinating lesions may be due to

intramyelinic edema or myelin vacuolation, which has been reported in toxic demyelination and some inborn errors of metabolism. Alternatively, myelin breakdown may reduce water movement in the extracellular space because of reduced fiber tract organization. Another potential mechanism is the presence of a hypercellular inflammatory infiltrate at the edge of acute demyelinating lesions or the presence of iron-laden macrophages, which can result in an overall lack of DWI signal because of very rapid T2* relaxation, as also seen in macrophages in the center of abscesses DWI can detect acute MS lesions as primary inflammatory changes in MS, consist of the cytotoxic type of edema followed by vasogenic edema. Combination of DWI and contrast enhanced MRI vield more positive results in diagnosis of acute MS attack [19]. Usually MS plaques have increase ADC values due to vasogenic edema as compared to contralateral white matter.

Leigh syndrome, also known as subacute necrotising encephalomyelopathy (SNEM),is a mitochondrial disorder with progressive neurodegeneration that invariably leads to death, usually in childhood.Typically, symptoms become evident before the age of 2, with the presentation in later childhood (juvenile form) or adulthood (adult form) being uncommon. Chronic energy deprivation leads to histological features such as [20] spongiform degeneration,capillary

proliferation, demyelination, neuronal loss and gliosis. These findings are similar to those seen in infarction

[21].

PML is typically seen in immunocompromised patents. The central portion of PML appears hyperintense on ADC due to vasogenic edema, while its peripheral rim appears hypointense on ADC due to cytotoxic edema. This helps in differentiating other pathologies. A study was done in 2009 by Straus Farber R, et al. to compare diffusion weighted imaging in gray and white matter brain regions of patients diagnosed with multiple sclerosis (MS) to those diagnosed with secondary demyelinating diseases such as acute disseminated encephalomyelitis (ADEM). Scans were performed and apparent diffusion coefficients of 12 regions of interest were determined in MS patients and ADEM patients[22]. They concluded that elevated apparent diffusion coefficients within the corpus callosum on diffusion weighted imaging may potentially help differentiate between patients with MS and patients with other diseases affecting the central nervous system white matter. Our findings corroborate with previous studies.



Fig-20:A case of Tumefactive Demyelination showing multiple well defined lesions in the subcortical white matter appearing hypointense peripherally with central hyperintensity on T2WI (A), showing "Open Ring" Enhancement of the active margins of the plaques on post contrast images(B), with restriction of margins on DWI (C) with low ADC values on ADC (D).



Fig-21: A case of Progressive Multifocal Leucoencephalopathy in an immunocompromised patient showing hyperintense lesions in right brachium Pontius extending to right cerebral hemisphere on FLAIR (A), showing restriction on DWI (B).



Fig-22:A case of Leigh's disease showing bilateral hypointense lesions in Lentiform nuclei on T1WI (A), appearing hyperintense on T2WI (B), showing restriction on DWI (C), with low ADC values (D)

Five patient of traumatic brain injury was studied in this group. The lesions were identified on DWI as well as ADC maps. In DAI the number of lesions identified on DW imaging was more as compared to the T2W imaging. All the lesions showed increased diffusion and loss of signal on ADC maps which correlated well with the previous study (Figure 23). Conventional MRI sequences tent to underestimate the true extend of DAI as shown injury occurs at the cellular level. The ADC is often found to decrease in patent with acute axonal shear injury signifying reduced water diffusion. DWI usually depicts additional shear

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injuries not visible on conventional, FLAIR, T2* image, GRE. Correlation exists between number of lesions on DWI and clinical outcome indicating that DWI is an important sequence in detecting DAI. The exact mechanism of restricted diffusion of DAI on DWI is still not fully understood, several explanations given are trauma induced brain ischemia and the process of trauma induced exotomy with the formation of retraction balls and concommitent cytoskeletal collapse along the severed axons [23].

A study was done by Liu AY *et al.* In 1999 on nine patients ranging in age from 26 to 78 years and were examined with conventional MR imaging (including fast spin-echo T2-weighted, fluid-attenuated inversion-recovery, and gradient-echo sequences) as well as echo-planar diffusion-weighted MR imaging 1 to 18 days after traumatic injury [24].

They concluded that decreased ADC can be demonstrated in patients with DAI in the acute setting and may persist into the subacute period, beyond that described for cytotoxic edema in ischemia.



Fig-23: A case of diffuse axonal injury in splenium of corpus callosum appearing hyperintense on T2WI (A), showing restriction on DWI (B) with corresponding low ADC values (C).

In the nine cases of epidermoid cyst studied in this group all showed restriction of diffusion on DW images with loss of signal on ADC maps. The other cases of cavernous hemangioma showed no restriction of diffusion while appearing bright on T2W images and were suppressed on FLAIR images and correlated well with the previous studies done. DWI sequence is useful for differentiation from arachnoid cysts due to increased signal (due to a combination of true restricted diffusion and T2 shine through) which is not seen with arachnoid cysts (Figure 24).

The low ADC values of ECs are the result of the dense keratinous and proteinous content of the cysts which limit the free protons causing hyperintensity on trace DW images [9].



Fig-24: A case of epidermoid cyst in at right CP angle cistern extending to right parapontine , prepontine ,right ambient and quadrigeminal cisterns appearing hypointense eon FLAIR (A), hyperintense on T2WI (B), showing restriction on DWI (C), with low ADC values (D).

In a study conducted by Annet L *et al.* in 2002 on six surgically proven epidermoid cysts patients in whom ADC calculation from T2-weighted DW-EPI-SE data were performed within the ECs and within the deep white matter and cerebrospinal fluid (CSF) as references concluded that all ECs displayed highest signal intensity on the DW trace images[25]. On ADC maps, epidermoids are markedly hypointense to CSF and are iso or slightly hyperintense to gray and white matter; whereas arachnoid cysts are markedly hyperintense with ADCs similar to those of CSF. On spin echo DW MR Images, epidermoid tumors frequently are

hyperintense, whereas arachnoid cysts are hypointense (isointense to CSF).

The study did have some limitations / drawbacks. Follow up imaging was not available in many of the patients. The histopathological diagnosis or clinical follow up was not available in some cases. There are too many subgroups within the present study and there are not sufficient numbers of patients in many subgroups. Hence it was not possible to draw conclusion in each and every subgroup. However maximum efforts have been made in correctly interpreting DW MR Images and ADC maps along with MR sequences and also along with clinical details of the patient.

CONCLUSION

DWI sequence is extremely useful in detection, etiopathogenesis and prognosis of intracranial lesions like infarcts, abscess, tumors, demyelination .The DW MR is a very useful sequence in evaluating stroke like symptoms. It can detect hyperacute infarcts within its window period i.e. 6 hours. In patients with multiple infarcts diffusion weighted imaging can identify the acute lesions. DWI is useful in early detection of HIE, when conventional MRI sequences are normal. DWI is extremely useful in detection of cvtotoxic edema in acute cortical laminar necrosis. DWI is useful in detection of atypical MRI findings of PRES which show cytotoxic edema. DW MR is not very useful to grade tumors but may serve as a guide to a highly cellular area for stereotactic biopsy. Also tumor margins are not clearly delineated on diffusion weighted imaging as compared to T1 and T2W imaging. Highly cellular tumors show low ADC values. DWI is very useful in differentiating the epidermoid from arachnoid cysts which may not be possible on conventional MRI sequences. IN CVST, DWI can separately evaluate vasogenic edema, reversible cytotoxic edema and cytotoxic edema due to venous infarction. DWI is useful in detection of encephalitis. DWI is useful in detection of cerebral abscess and differentiating it from tumors. In case of demyelinating diseases, by showing peripheral diffusion restriction DWI can identify an active lesion and can help in prognostication and response to treatment. In cases of trauma especially DAI, diffusion weighted imaging may identify more lesions compared to conventional MR sequences which is useful for prognostification and patient management.

REFERENCES

- Osborn AG. Infections, white matter abnormalities and degenerative diseases. In: Osborn AG. Diagnostic neuroradiology. 1st ed. Banglore: Harcourt brace & company Asia PTE LTD; 1997. p. 673-715.
- Stejskal EO, Tanner JE. Spin diffusion measurements: spin echoes in the presence of a time-dependent field gradient. The journal of chemical physics. 1965 Jan 1;42(1):288-92.
- Baliyan V, Das CJ, Sharma R, Gupta AK, Diffusion weighted imaging: Technique and applications. World J Radiol. 2016 Sep 28;8(9):785-798.PMID: 27721941 PMCID: PMC5039674.
- Bryan RN, Levy LM, Whitlow WD, Killian JM, Preziosi TJ, Rosario JA. Diagnosis of acute cerebral infarction: comparison of CT and MR imaging. American Journal of Neuroradiology. 1991 Jul 1;12(4):611-20.
- Kon Chu MD, Diffusion Weighted Magnetic Resonance in cerebral venous thrombosis, Arch Neurol. 2001;58-1569:1576.

- Kidwell CS, Alger JR, Di Salle F, Starkman S, Villablanca P, Bentson J, Saver JL. Diffusion MRI in patients with transient ischemic attacks. Stroke. 1999 Jun 1;30(6):1174-80.
- 8. Greenfield JG, Adams JH, Corsellis JA, Duchen LW. Greenfield's neuropathology. 1984.
- Bryan RN, Willcott MR, Schneiders NJ, Ford JJ, Derman HS. Nuclear magnetic resonance evaluation of stroke. A preliminary report. Radiology. 1983 Oct;149(1):189-92.
- Kertesz A, Black SE, Nicholson L, Carr T. The sensitivity and specificity of MRI in stroke. Neurology. 1987 Oct 1;37(10):1580-.
- 11. Hoehn-Berlage M. Diffusion-weighted NMR imaging: application to experimental focal cerebral ischemia. NMR in Biomedicine. 1995 Nov 1;8(7):345-58.
- Moseley ME, Butts K, Yenari MA, Marks M, Crespigny AD. Clinical aspects of DWI. NMR in Biomedicine. 1995 Nov 1;8(7):387-96.
- 13. Chan JH, Tsui EY, Chau LF, Chow KY, Chan MS, Yuen MK, Chan TL, Cheng WK, Wong KP. Discrimination of an infected brain tumor from a cerebral abscess by combined MR perfusion and diffusion imaging. Computerized medical imaging and graphics. 2002 Jan 2;26(1):19-23.
- 14. Sugahara T, Korogi Y, Kochi M, Ikushima I, Shigematu Y, Hirai T, Okuda T, Liang L, Ge Y, Komohara Y, Ushio Y. Usefulness of diffusionweighted MRI with echo-planar technique in the evaluation of cellularity in gliomas. Journal of Magnetic Resonance Imaging. 1999 Jan 1;9(1):53-60.
- Klisch J, Husstedt H, Hennings S, Velthoven VV, Pagenstecher A, Schumacher M. Supratentorial primitive neuroectodermal tumours: diffusionweighted MRI. Neuroradiology. 2000 Jun 1;42(6):393-8.
- 16. Ebisu T, Tanaka C, Umeda M, Kitamura M, Naruse S, Higuchi T, Ueda S, Sato H. Discrimination of brain abscess from necrotic or cystic tumors by diffusion-weighted echo planar imaging. Magnetic resonance imaging. 1996 Jan 1;14(9):1113-6.
- Chang SC, Lai PH, Chen WL, Weng HH, Ho JT, Wang JS, Chang CY, Pan HB, Yang CF. Diffusion-weighted MRI features of brain abscess and cystic or necrotic brain tumors: comparison with conventional MRI. Clinical imaging. 2002 Aug 31;26(4):227-36.
- Kim YJ, Chang KH, Song IC, Kim HD, Seong SO, Kim YH, Han MH. Brain abscess and necrotic or cystic brain tumor: discrimination with signal intensity on diffusion-weighted MR imaging. AJR.

^{6.} E. Ralph Heinz, James M. Provenzale, Imaging Findings in Neonatal Hypoxia: A Practical Review, AJR:192, January 2009.

Available online at https://saspublishers.com/journal/sjams/home

American journal of roentgenology. 1998 Dec;171(6):1487-90.

- Warmuth-Metz M, Hofmann E, Büsse M, Solymosi L. Uncommon morphologic characteristics in Leigh's disease. American journal of neuroradiology. 1999 Jun 1;20(6):1158-60.
- 20. Cavanagh JB, Harding BN. Pathogenic factors underlying the lesions in Leigh's disease. Tissue responses to cellular energy deprivation and their clinico-pathological consequences. Brain. 1994;117 (Pt 6) (6): 1357-76.
- Liu AY, Maldjian JA, Bagley LJ, Sinson GP, Grossman RI. Traumatic brain injury: diffusionweighted MR imaging findings. American Journal of Neuroradiology. 1999 Oct 1;20(9):1636-41.
- 22. Straus Farber R1, Devilliers L, Miller A, Lublin F, Law M, Fatterpekar G, Delman B, Naidich T.Differentiating multiple sclerosis from other causes of demyelination using diffusion weighted imaging of the corpus callosum. J Magn Reson Imaging. 2009 Oct;30(4):732-6.PMID: 19787715
- 23. Ezaki Y, Tsutsumi K, Morikawa M, Nagata I. Role of diffusion-weighted magnetic resonance imaging in diffuse axonal injury. Acta Radiologica. 2006 Sep;47(7):733-40.
- Ball T, Malik O, Roncaroli F, Quest RA, Aviv RI. Apparent diffusion coefficient changes and lesion evolution in Balo's type demyelination-correlation with histopathology. Clin Radiol 2007;62:498 – 503.
- 25. Annet L, Duprez T, Grandin C, Dooms G, Collard A, Cosnard G. apparent diffusion coefficient measurements within intracranial epidermoid cysts in six patients. Neuroradiology 2002; 44(4): 326-8.